



THE UNIVERSITY *of* EDINBURGH

## Edinburgh Research Explorer

### Posttraumatic stress symptom severity, prevalence and impact in ambulance clinicians

**Citation for published version:**

Davis, K, Macbeth, A, Warwick, R & Chan, W 2019, 'Posttraumatic stress symptom severity, prevalence and impact in ambulance clinicians: The hidden extent of distress in the emergency services', *Traumatology*. <https://doi.org/10.1037/trm0000191>

**Digital Object Identifier (DOI):**

[10.1037/trm0000191](https://doi.org/10.1037/trm0000191)

**Link:**

[Link to publication record in Edinburgh Research Explorer](#)

**Document Version:**

Peer reviewed version

**Published In:**

Traumatology

**Publisher Rights Statement:**

©American Psychological Association, 2019. This paper is not the copy of record and may not exactly replicate the authoritative document published in the APA journal. Please do not copy or cite without author's permission. The final article is available, upon publication, at: <http://dx.doi.org/10.1037/trm0000191>

**General rights**

Copyright for the publications made accessible via the Edinburgh Research Explorer is retained by the author(s) and / or other copyright owners and it is a condition of accessing these publications that users recognise and abide by the legal requirements associated with these rights.

**Take down policy**

The University of Edinburgh has made every reasonable effort to ensure that Edinburgh Research Explorer content complies with UK legislation. If you believe that the public display of this file breaches copyright please contact [openaccess@ed.ac.uk](mailto:openaccess@ed.ac.uk) providing details, and we will remove access to the work immediately and investigate your claim.



1  
2 Post-Traumatic Stress Disorder (PTSD) is reported to be more prevalent in ambulance  
3 clinicians than the general population. Given the high frequency of exposure to high stress  
4 and traumatic situations over the course of an ambulance clinician's career, the current study  
5 examined the prevalence of post-traumatic stress (PTS) symptoms and the severity of distress  
6 related to these symptoms in this population. A total of 508 ambulance clinicians, including  
7 paramedics and technicians, completed the Life Events Checklist-Five and the Impact of  
8 Events Scale-Revised. Severity of distress associated with PTS symptoms was determined by  
9 using commonly used clinical cut-off scores. Nearly 50% of ambulance clinicians reported  
10 distress arising from symptoms of PTS of severity sufficient to be of clinical concern. Over  
11 23% reported severe levels of distress. Results indicate concerning levels of distress relating  
12 to PTS within the ambulance service. Anchoring PTS to an index event and measuring  
13 duration of symptoms relative to that event is likely not accounting for the complex  
14 interaction of previous and further exposures on presentation. This may mask the extent of  
15 the impact of trauma exposure in populations with recurrent exposure to distressing  
16 situations.

17 **Key Words: Post-traumatic stress, emergency services, ambulance clinician, emergency**  
18 **medical technician, paramedic**

19 Exposure to highly distressing and traumatic situations has the ability to impact on  
20 individuals long after the time of the event (McFarlane, 2010) and has consistently been  
21 related to mental health problems (Petrie et al., 2018), in particular Post-Traumatic Stress  
22 Disorder (PTSD; Yehuda et al., 2015). Following exposure to a distressing event (death,  
23 violence, serious injury) the Diagnostic and Statistical Manual of Mental Disorders- Fifth  
24 edition (American Psychiatric Association [APA], 2013) defines PTSD as symptoms of re-  
25 experiencing the event(s); avoidance of trauma-related stimuli; negative thoughts that have

worsened since the exposure; and trauma related arousal and reactivity that began or worsened after the exposure. These post-traumatic stress (PTS) symptoms have to last for at least a month and cause significant distress or functional impairment. Acute Stress Disorder (ASD) is similar to that of the criteria for PTSD; however, symptoms are present for under a month (APA, 2013).

Ambulance clinicians are often exposed to highly distressing situations as a function of their working life. Increasingly, evidence suggests that the impact of chronic job stressors in the ambulance service leads to increased levels of mental health problems and physical problems (Hegg-Deloye et al., 2014). Fifty-four percent of staff in the Scottish Ambulance Service reported that they believed work pressure has affected their health (BBC News, 2017).

Ambulance clinicians also demonstrate higher prevalence rates of PTSD compared to other populations. Amongst UK ambulance service workers, PTSD prevalence has been found to be approximately 22% (Bennett, Williams, Page, Hood, & Woollard, 2004), six to eight times greater than the prevalence rate found in the general population (2.6%-3.3%; National Institute for Health and Care Excellence [NICE], 2016). Further, compared with other emergency services, ambulance personnel show the highest levels of PTSD (Berger et al., 2012) and a recent meta-analysis indicated prevalence of PTSD amongst ambulance clinicians is estimated to be 11% (Petrie et al., 2018).

Inclusion criteria for studies focusing on the prevalence of PTSD and associated distress generally rely on diagnostic conditions being met. Symptoms are anchored to an index event that occurred at least four weeks prior to the time of study and symptoms are reported to last for at least four weeks, or less than four weeks for ASD. Often the index event is identified by asking participants to select the worst or most upsetting event and PTS symptoms are assessed in relation to that single event (Kessler et al., 2017). By doing so an assumption is made that it is valid to assume that the presence of PTS symptoms and distress at that time

are related to one single event and that this relationship exists in isolation from other variables. However, studies have demonstrated that the probability of developing PTS increases with exposure to multiple traumatic events (Kilpatrick et al., 2013). It has been established that individuals with sub-threshold PTSD are at risk of developing more severe symptoms with further exposure and environmental stress (McFarlane, 2010). Presence of PTS symptoms that are not severe enough to meet threshold criteria for PTSD are also related to significant levels of distress and impairment (McLaughlin et al., 2015). Evidence also indicates PTSD can be a chronic illness often with individuals experiencing a waxing and waning of symptoms over time (Ozer, Best, Lipsey, & Weiss, 2003).

Clearly the evidence base demonstrates that PTS trajectories are varied, with people presenting with symptoms at different time points and of varying durations. These variations highlight a limitation in using diagnostic criteria in cross-sectional research in populations with high exposure rates to distressing situations. Due to the high rate of exposure there are several possible trajectories of PTS that can result in the presence of symptoms considered clinically severe. The extent to which these symptoms and associated severity/distress are captured in research will therefore likely vary depending on methodology and how the presence of symptoms are anchored to an index event in accordance with discrete diagnostic classification. Critically, it may not necessarily reflect an accurate picture of current PTS severity if participants are asked to identify the worst or most upsetting event and provide a profile of symptom severity exclusively to that event. This is because while one event may be identified as the worst event an individual may not identify or perceive all of their current symptoms as directly caused by that event. As participants are instructed to describe their symptoms in relation to that event alone they may report lower levels of PTS than what they are currently experiencing, as in reality, other exposures are also contributing to overall severity (Figure 1). Given this possibility, it is likely the methodology of the study would

determine diagnosis due to what is captured as the index event at the time of study. Thus these types of studies in high exposure populations are vulnerable to excluding presentations that are clinically severe and significantly distressing because they are not accounting for the interactions between PTS symptoms and multiple event exposure.

*>Insert Figure 1.*

*Figure 1.* Theoretical illustration of PTS symptom severity trajectory anchored to specific events versus actual trajectory of symptoms.  $t_0$ - $t_2$ , time of event exposure;  $t_{\text{study}}$ , time of study; dashed line represents trajectory of symptoms anchored to specific event; solid line represents actual trajectory of symptoms.

In conjunction, this highlights a particular weakness in the literature in regard to emergency services: research tends to examine PTSD as a discrete episode rather than continuous, with a focus on individuals who meet diagnostic criteria with reference to a single event and a four week duration of symptoms anchored specifically to that event. These studies may consequently be significantly influenced by the time of the study, time of index event selected and the supposition that duration and severity of symptoms are exclusively related to that event. This does not account for the potential effect of prior event exposures, presence of PTS preceding the index event identified and the fluid nature of symptom severity. Therefore, it is possible that research is not capturing the extent of PTS and associated distress present in these services when restricted to only including individuals based on discrete diagnostic criteria.

It is important to understand the extent of current distress in emergency services not only in terms of the individual's wellbeing, but also because evidence highlights the potential mediating effect acute stress has between PTSD and performance on complex cognitive tasks and decision making (Regehr & LeBlanc, 2017). The reality for many emergency service personnel is that they are often working in situations that require a high cognitive demand in times of severe stress, where one could conceptualise traumatic stressors being at the severe end of this spectrum (Bremner, 2006). From a neurobiological perspective, evidence increasingly demonstrates that acute and chronic stress has an atrophying effect on the neural circuit that allows one to engage in flexible thinking, decision making, planning and goal-orientated behaviour (hippocampus-prefrontal cortex [PFC] neural circuit; de Quervain, Schwabe, & Roozendaal, 2017). This neural circuit plays a key role in inhibiting the amygdala (Akirav & Maroun, 2007), the brain structure that orchestrates a survival response by engaging the stress response system (Rodrigues, LeDoux, & Sapolsky, 2009). The presence of stress critically appears to produce a reliance towards a more rigid cognitive style reliant on habit based memory systems (Ness & Calabrese, 2016) and weakens the PFC's hold on the amygdala (Akirav & Maroun, 2007; Arnsten, 2009).

Research indicates that individuals with PTS show increased activation in the stress response system and exhibit dysfunction in the amygdala-hippocampus-PFC neural circuit (Bremner, 2006; Liberzon & Abelson, 2016). It is generally considered that the deficits in this neural circuit give rise to observable symptoms of PTSD (Elzinga & Bremner, 2002; Liberzon & Abelson, 2016). Thus evidence suggests that ongoing PTS and stress can sustain the cycle of atrophy of key neural circuits required for inhibiting the stress response system and allowing for a more flexible response to situations (Arnsten, 2009; Bremner, 2006; Radley et al., 2004). Moreover, it indicates a potentially greater risk of people being cognitively impacted by their experiencing distress consequent on their symptoms of PTS.

A further concern this raises is the susceptibility of individuals with PTS to develop further fear based associations upon exposure to high stress conditions. The activation of the stress response system enhances learning of conditioned fear associations and appears to reduce the ability to extinguish such associations (Akirav & Maroun, 2007; Elzinga & Bremner, 2002). While this fear association is unlikely to be unlearned, a new association can be learned to compete and overlay the initial association, likely via the inhibitory effect of the PFC on the amygdala (Craske, Treanor, Conway, Zbozinek, & Vervilet, 2014; Milad et al., 2009). However, as the original association still exists, it is susceptible to being re-established as the primary association and the affiliated fear response can re-emerge (Craske et al., 2014). Critically, one such catalyst for re-establishment is further exposure to adverse events (Craske et al., 2014). Consequently, PTS symptoms have potential to fluctuate in severity in this population due to the likelihood of encountering further traumatic and high stress situations. Moreover, it highlights the possibility that people who already experience PTS will be more vulnerable to increased generalisation of symptoms to further events as fear based associative learning is enhanced at time of exposure.

Finally, the advancement in understanding of the neurobiology of the encoding and retrieval of trauma memories demonstrates a further issue with research that relies on anchoring symptoms to an index event in these populations. In response to high stress situations, cortisol and norepinephrine are released into the brain (Elzinga & Bremner, 2002). Evidence suggests that the impact of cortisol on the hippocampus can impair formation of explicit memory, while norepinephrine increases the formation of the implicit coding of fear memory via the amygdala (Elzinga & Bremner, 2002; Ness & Calabrese, 2016). Explicit memory is a form of memory recall whereby events and facts are consciously remembered. Implicit memory does not require conscious attention during encoding or retrieval and can be defined as affective knowledge, bodily sensations and behavioural response patterns of an event

without conscious memory (Elzinga & Bremner, 2002). A neurobiological perspective of PTS is that the aspects of the traumatic experience are encoded implicitly and are not integrated into explicit memory. That is, at the time of the event at least some of the aspects of the experience are not encoded into explicit memory, and therefore are not integrated into the autobiographical memory of the individual. As the memory is implicitly encoded an individual may remember this experience in the form of sensations, emotions and behaviours without being able to explicitly recall or anchor this event as being in the past (Elzinga & Bremner, 2002). For populations where high stress experiences are more likely to occur on a frequent basis this produces obvious limitations on research seeking to anchor symptoms to a single event. An assumption is made that the individual can connect feelings, sensations and behavioural reactions to a previous individual event; in effect this is assuming these experiences have been integrated into explicit memory. However, evidence would suggest that it is because these experiences are not integrated into explicit memory symptoms of PTS arise.

The field has significantly advanced in its understanding of the impact of exposure to highly distressing situations and the impact of high levels of stress on key neural circuits and how this presents on a phenotypic level. Exposure to recurrent distressing and high stress situations provides an environment for symptom severity variation over time; likely influenced by the complex multidirectional interaction between multiple exposures, PTS, stress and other environmental pressures. Given the distressing nature of PTS symptoms and the effect on cognitive systems, it is important to capture the prevalence of PTS and severity of distress as a consequence of their symptoms experienced by ambulance clinicians. The aim of this study is to investigate the prevalence of PTS symptoms and the severity of distress related to these symptoms with due regard to the dynamic nature of PTS trajectories. It is hypothesised that by removing the diagnostic criteria of duration of symptoms and the



anchoring of presenting symptoms to an index event that the prevalence of symptom severity and distress will be higher than that reported in diagnostically grounded studies.

## **Method**

### **Participants**

Participants were ambulance clinicians recruited via an invitation email sent out nationwide to the Scottish Ambulance Service (SAS). Participants had to be over 18 years of age and employed within the SAS either as a qualified ambulance technician or as a paramedic.

### **Measures**

**The Life Events Checklist-5 (LEC-5;** Weathers et al., 2013). The LEC-5 is a self-report screening tool used to assess exposure to potentially traumatic aetiological experiences. It contains 16 items of common traumatic events related to PTSD and requires participants to answer under what context they were exposed to the event (e.g. ‘Happened to me’, ‘Part of my Job’). The LEC-5 is an updated version of the LEC which demonstrates a strong correlation with PTS and convergent validity with other measures of traumatic exposure (Gray, Litz, Hsu, & Lombardo, 2004). The LEC-5 was used to verify that each participant had at least one exposure to trauma. Cronbach’s alpha for the current sample was .75.

**The Impact of Events Scale- Revised (IES-R;** Weiss, 2007). The IES-R is a 22-item measure used to assess psychological responses following stressful life events, measuring symptoms of hyperarousal, intrusion and avoidance. Participants were asked to indicate how distressing they had found each symptom listed during the last seven days. On a 5-point Likert Scale participants ranked answers ranging from ‘Not at all’ to ‘Extremely’. It has

demonstrated a high internal consistency with a Cronbach's alpha of .96, good test-retest reliability and a single factor solution (Creamer, Bell, & Failla, 2003). Cronbach's alpha in the current sample was 0.95 for the total scale, .88 for the hyperarousal sub-cluster, .92 for the intrusions sub-cluster and .84 for the avoidance sub-cluster.

## **Procedure**

Ethics approval was obtained from the University of Edinburgh and the SAS Research and Development Board. An invitation email was sent to employees of the SAS providing information regarding the study and a secure web link to the questionnaire. An advertisement was also placed on the SAS intranet inviting paramedics and ambulance clinicians to complete the survey. The survey could only be accessed via the link provided. In order to complete the study participants had to select if they were a paramedic or an ambulance technician.

The information sheet outlined the purpose of the study and written consent was sought prior to the administration of a battery of psychometric scales. The questionnaires were completed online as part of a battery of psychometric scales within a single session and all completed surveys were anonymous. The order of the questionnaires was presented as listed in the measures section starting with the LEC-5, immediately followed by the IES-R. A de-briefing sheet was provided upon completion where participants were provided information about speaking to their GP if they felt distressed, as well as the number for the counselling service provided by the SAS.

## **Statistical Analysis**

Clinical cut-off scores recommended in the literature were used to assess the severity of distress associated with PTS symptom presence. A score of 24 and above indicated severity

of clinical concern, 33 and above indicated a probable diagnosable level of PTSD or ASD (if other relevant criteria had been met) and a score over 37 indicated severe distress (Asuaki et al., 2002; Creamer et al., 2003; Kawamura, Kim, & Asukai, 2001). Multivariate normality was tested by checking skewness and kurtosis. All variables were normally distributed. Bivariate correlations were used to examine the relationship between age and years of experience with total IES-R score. A secondary analysis was conducted to investigate if there was a significant difference in IES-R sub-scale scores measuring hyperarousal, avoidance and intrusion symptoms. A within-subjects ANOVA was used to compare the mean scores across the three variables. Further analysis of the subscale mean score differences were carried out using paired sample t-tests

## Results

A total of 508 ambulance Clinicians were recruited consisting of paramedics ( $N=355$ , 69.9%) and ambulance technicians ( $N=153$ , 30.1%). The age range was from 21 to 62 ( $M=44.97$ ,  $SD=9.06$ ). The years of experience ranged from under a year to 39 years ( $M=14.69$ ,  $SD=9.22$ ). In total 342 (67.3%) of the participants were male, 160 were female (31.5%), and 6 preferred not to say (1.2%). All participants reported exposure to at least one traumatic event, either via experiencing it first hand, witnessing it or learning about it happening to a close family or friend over their lifetime. There are approximately 2800 ambulance clinicians employed by the SAS (SAS personal communication), therefore the overall response rate was approximately 18%. There was no missing data.

Results from the descriptive analysis of the IES-R showed that 51.8% of ambulance clinicians did not have any significant symptoms, 14.6% showed levels of distress associated with symptoms of clinical concern, 10.0% showed probable diagnosable levels of distress and 23.6% showed severe distress when asked about symptoms over the past week.

Age and total IES-R score were not significantly correlated ( $r = -.00, p = .84$ ). Years of experience and total IES-R scores were not significantly correlated ( $r = .04, p = .38$ ). A significant difference was found between the mean sub-scale scores of intrusions ( $M = 1.14, SD = .82$ ), hyperarousal ( $M = .94, SD = .89$ ) and avoidance ( $M = 1.28, SD = .89$ ),  $F(1.91, 969.35) = 88.18, p < .001$ . There was a significant difference between avoidance and intrusion scores,  $t(507) = -5.33, p < .001, d = .17$ . There was a significant difference between avoidance and hyperarousal scores,  $t(507) = 7.22, p < .001, d = .38$ . There was a significant difference between intrusion and hyperarousal scores,  $t(507) = 14.91, p < .001, d = .23$ . Figure 2 shows how the sample of ambulance clinicians completed the scale for each item on the IES-R.

*>Insert Figure 2.*

Figure 2. Overall level of distress reported per item on the IES-R.

## Discussion

The aim of the current study was to establish how severe ambulance clinicians' experiences of distress are as a result of their reporting trauma symptomatology. Overall, this research shows a considerable level of severe distress relating to PTS. Approximately half the population demonstrated levels of PTS indicative of clinical concern, with around 24% of the population presenting with severe levels. Whilst previous research has established that over one in five ambulance clinicians have clinically diagnosable levels of PTSD (Bennett et al., 2004) the current study shows that one in two suffer notable symptoms. This indicates a significant level of distress present at any given time in this population when diagnostic limitations are removed, and supports the practical need to rethink conceptualisation of PTS to ensure adequate support is provided to emergency workers.

Overall distress related to hyperarousal symptoms was significantly lower compared to symptoms of intrusion and avoidance. This disparity may be best understood by taking the unique context in which ambulance clinicians are exposed to trauma. Ambulance clinicians likely need to remain in a state of alertness and preparedness throughout a shift. Given this environmental demand, maintaining a heightened state of arousal may be more likely to be viewed as normal and adaptive, and moreover, at an optimal level may also be advantageous within the context of the environment. Thus, relatively lower distress associated with hyperarousal could be related to the appropriateness of increased levels of arousal within the environmental framework. Therefore, it is possible that it may not necessarily be that ambulance clinicians experience less severe symptoms of hyperarousal compared to intrusions and avoidance; rather, distress associated with intrusion and avoidance symptoms might be more readily recognised as they are to a greater extent incongruent with the environmental norm. Further research however, would be needed to explore this as a possibility. While there was a significant difference between intrusion and avoidance symptom severity the effect size was small. Therefore, it is proposed that this difference is probably relatively inconsequential between the subscales.

### **Clinical and Research Implications**

Regardless of whether PTSD criteria are met, our finding that almost half of the population suffering with PTS symptoms in an emergency service is of note. A minimum duration of symptoms of four weeks or more anchored to an index event may mask important clinical findings that could benefit those in these occupations. Moreover, it likely camouflages the true impact of working in a profession where trauma is an intrinsic part of the job. The high number of participants falling into the category of clinical concern and above is possibly related to recurrent exposure to stressful situations that are cognitively demanding against further environmental pressures, such as response times and high demand.

The presence of PTS symptoms and associated distress is indicative of an active stress response system and impairment in the neural circuit required for inhibition of this system (Akirav & Maroun, 2006; Liberzon & Abelson, 2016). Thus, there is potential for an increased risk of establishment of further fear conditioned associations, alongside a decreased ability to overlay these associations with a non-threat association (Akirav & Maroun, 2006; Elzinga & Bremner, 2002). This has obvious repercussions for the efficacy of treatment within this population. Cognitive Behaviour Therapy and Eye Movement Desensitisation and Reprocessing are the first line treatment for PTSD (NICE, 2018), and whilst each postulate different cognitive mechanisms of change, both interventions likely rely on the use of the PFC-Hippocampus neural circuit to reduce symptoms. Evidence increasingly suggests there is likely a context processing deficit in PTSD (Liberzon & Abelson, 2016); the presence of PTS in the absence of actual threat is indicative of a neural failure to recognise or contextualise that the current environment is not threatening. However, in populations with high exposure rates to high stress conditions there is likely not such a clear dichotomy. The current context in which symptoms are present may in reality be highly stressful, and thus the environment itself is potentially not facilitative to recovery from these symptoms.

Given the significant level of distress related to PTS in this population future research should take into account the bidirectional impact of further traumatic/high stress exposure and PTS. This should focus on severity of expression of PTS, and moreover, given the susceptibility of symptom relapse post treatment due to further event exposures (Craske et al., 2014) focus on optimising long term treatment outcomes. Such research may also allow for the development of treatments that specifically address symptoms that likely interplay with multiple traumatic events and other key environmental variables. One such possibility may be to provide non-discrete episodes of intervention, whereby PTS specific treatments sit complimentary to a more dynamic formulation of PTS. Critically, the neurobiological impact of ongoing stress on

treatment efficacy should be taken into account: an active stress response system in a context of an environment that is highly stressful may result in fear based associations being more easily established, increased difficulty in establishing an inhibitory overlay association and decreased ability to integrate experiences into explicit memory. Along this line, there may be benefit in interventions that promote adaptive strategies for coping with high levels of stress on an individual level, as well as adaptations within the system that reduce other environmental pressures. From a neurobiological perspective this may promote strengthening of PFC connectivity to the amygdala and a reduction of environmental variables that trigger the stress response system respectively. Further, if stress and PTS result in a reliance on more habitual based memory systems, then the use of advancing technology such as virtual reality may prove invaluable in emulating as much as possible the situations that are likely to be most overwhelming and relatively unpredictable in nature, for example, multiple casualties, terrorist related threats.

## **Limitations**

All data was collected via self-reported measures and thus may be subject to biases, particularly the tendency for self-report measures to result in modestly higher reporting of symptoms compared to interview (Bowling, 2005). However, whilst clinical interviews demonstrate the most reliable form of assessment, screening measures serve as a less time-intensive, resource-laden indicator of PTS severity across a population (Coffey, Gudmundsdottir, Beck, Palyo, & Miller, 2006) and have likely allowed the recruitment of the uncommonly large sample here. Further, there is potential for a selection bias in the sample which allows for the possibility for the results presented here to be skewed in either direction from the true value of distress in the population. It could be that response rates from those who experience high levels of distress could be reduced due to the general tendency to avoid reminders of distress, a common trait in PTS. It could also be that those who are unaffected

by exposure to stressful situations did not feel the need to complete the questionnaire and thus could be under-represented. However, at its most conservative, approximately ten percent of the whole population reported presenting with clinically concerning symptoms. Although, this would be lower than reports of PTSD prevalence found in other studies, such as Bennett et al. (2004), and thus is unlikely.

The IES-R does not capture functional impairment associated with severity; however, it is a measure unaligned to diagnostic classification systems, and thus does not have the same constraints of measures that do. Additionally, while this study highlights the relevance of investigating PTS in the context of multiple exposures and removal of four week duration of symptoms as an inclusion criteria, there is no direct comparison between this and diagnostic criteria for PTSD in this study. This study highlights the need for longitudinal research focusing on PTS trajectories, where assessment of severity is not based on a discrete diagnostic framework with symptoms only counted and recognised as relating to a single event.

## **Conclusions**

Anchoring PTS to an index event and measuring duration of symptoms relative to that event is likely not accounting for the complex interaction of previous and succeeding exposures on presentation. By not taking into account the dynamic, interactive and systemic nature of PTS there is likely a failure to capture the true extent of PTS in high exposure populations. Research is continually advancing our understanding of PTS, neurobiological underpinnings and the likely entangled and complex interactions of these symptoms and how they interweave with further trauma exposure and other environmental pressures. Given this, and the considerable level of distress reported here, it may be time to reconceptualise how we measure the impact of trauma exposure in these populations. Perhaps we should move past



the current constraints of measuring PTS within a discrete episodic framework, as it may not reflect the true extent of the burden of trauma exposure in these populations. Further research is needed to map out PTS over time in populations with recurrent exposures that are not reliant on assessing PTS from methodologies orientated to single event assessment. By conducting research in these populations that is consistent with the neurobiological understanding of the impact of stress and trauma we can develop a parsimonious conceptualisation of PTS and its relation to the environment it presents in. Critically, with this advancement interventions can be developed and adapted to adequately complement such a framework.

### Acknowledgements

We would like to thank the Scottish Ambulance Service, in particular, Dr David Fitzpatrick, as well as all the participants, for their support of the study.

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

### References

- Akirav, I. & Maroun, M. (2006). Ventromedial prefrontal cortex is obligatory for consolidation and reconsolidation of object recognition memory. *Cerebral Cortex*, 16(12), 1759–1765. <http://doi.org/10.1093/cercor/bhj114>
- Akirav, I. & Maroun, M. (2007). The role of the medial prefrontal cortex-amygdala circuit in stress effects on the extinction of fear. *Neural Plasticity*, 2007. <http://doi.org/10.1155/2007/30873>
- American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders*. 5th edition. <http://doi.org/10.1176/appi.books.9780890425596.dsm07>
- Arnsten, A. F. (2009). Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience*, 10(6), 410. <http://doi.org/10.1038/nrn2648>

Asukai, N., Kato, H., Kawamura, N., Kim, Y., Yamamoto, K., Kishimoto, J., ... & Nishizono-Maher, A. (2002). Reliability and validity of the Japanese-language version of the impact of event scale-revised (IES-R): four studies of different traumatic events. *The Journal of Nervous and Mental Disease*, 190(3), 175–182. <http://dx.doi.org/10.1097/00005053-200203000-00006>

BBC News (2017). Scottish Ambulance Service staff morale 'at rock bottom'. Retrieved from <http://www.bbc.co.uk/news/uk-scotland-42019454> 2017

Bennett, P., Williams, Y., Page, N., Hood, K., & Woollard, M. (2004). Levels of mental health problems among UK emergency ambulance workers. *Emergency Medicine Journal*, 21(2), 235–236. <http://doi.org/10.1136/emj.2003.005645>

Berger, W., Coutinho, E. S. F., Figueira, I., Marques-Portella, C., Luz, M. P., Neylan, T. C., ... & Mendlowicz, M. V. (2012). Rescuers at risk: a systematic review and meta-regression analysis of the worldwide current prevalence and correlates of PTSD in rescue workers. *Social Psychiatry and Psychiatric Epidemiology*, 47(6), 1001–1011. <http://dx.doi.org/10.1007/s00127-011-0408-2>

Bowling, A. (2005). Mode of questionnaire administration can have serious effects on data quality. *Journal of Public Health*, 27(3), 281–291. <http://doi.org/10.1093/pubmed/fdi031>

Bremner, J. D. (2006). Stress and brain atrophy. *CNS & Neurological Disorders-Drug Targets (Formerly Current Drug Targets-CNS & Neurological Disorders)*, 5(5), 503–512. <http://doi.org/10.2174/187152706778559309>

Coffey, S. F., Gudmundsdottir, B., Beck, J. G., Palyo, S. A., & Miller, L. (2006). Screening for PTSD in motor vehicle accident survivors using the PSS-SR and IES. *Journal of Traumatic Stress*, 19(1), 119–128. <http://doi.org/10.1002/jts.20106>

Craske, M. G., Treanor, M., Conway, C. C., Zbozinek, T., & Vervliet, B. (2014). Maximizing exposure therapy: an inhibitory learning approach. *Behaviour Research and Therapy*, 58, 10–23. <http://doi.org/10.1016/j.brat.2014.04.006>

Creamer, M., Bell, R., & Failla, S. (2003). Psychometric properties of the impact of event scale-revised. *Behaviour Research and Therapy*, 41(12), 1489–1496. <http://doi.org/10.1016/j.brat.2003.07.010>

de Quervain, D., Schwabe, L., & Roozendaal, B. (2017). Stress, glucocorticoids and memory: implications for treating fear-related disorders. *Nature Reviews Neuroscience*, 18(1), 7. <http://doi.org/10.1038/nrn.2016.155>

Elzinga, B. M., & Bremner, J. D. (2002). Are the neural substrates of memory the final common pathway in posttraumatic stress disorder (PTSD)? *Journal of Affective Disorders*, 70(1), 1–17. [https://doi.org/10.1016/S0165-0327\(01\)00351-2](https://doi.org/10.1016/S0165-0327(01)00351-2)

426 Gray, M. J., Litz, B. T., Hsu, J. L., & Lombardo, T. W. (2004). Psychometric properties of  
 427 the life events checklist. *Assessment*, 11(4), 330–341.  
 428 <http://doi.org/10.1177/1073191104269954>

429 Hegg-Deloye, S., Brassard, P., Jauvin, N., Prairie, J., Larouche, D., Poirier, P., ... & Corbeil,  
 430 P. (2014). Current state of knowledge of post-traumatic stress, sleeping problems,  
 431 obesity and cardiovascular disease in paramedics. *Emergency Medicine*  
 432 *Journal*, 31(3), 242–247. <http://doi.org/10.1136/emered-2012-201672>

433 Kawamura, N., Kim, Y., & Asukai, N. (2001). Suppression of cellular immunity in men with  
 434 a past history of posttraumatic stress disorder. *American Journal of Psychiatry*,  
 435 158(3), 484–486. <http://doi.org/10.1176/appi.ajp.158.3.484>

436 Kessler, R. C., Aguilar-Gaxiola, S., Alonso, J., Benjet, C., Bromet, E. J., Cardoso, G., ... &  
 437 Koenen, K.C. (2017). Trauma and PTSD in the WHO world mental health surveys.  
 438 *European Journal of Psychotraumatology*, 8(sup5), 1353383.  
 439 <http://doi.org/10.1080/20008198.2017.1353383>

440 Kilpatrick, D. G., Resnick, H. S., Milanak, M. E., Miller, M. W., Keyes, K. M., & Friedman,  
 441 M. J. (2013). National estimates of exposure to traumatic events and PTSD  
 442 prevalence using DSM-IV and DSM-5 criteria. *Journal of Traumatic Stress*, 26(5),  
 443 537–547. <http://doi.org/10.1002/jts.21848>

444 Liberzon, I., & Abelson, J. L. (2016). Context processing and the neurobiology of post-  
 445 traumatic stress disorder. *Neuron*, 92(1), 14–30.  
 446 <http://doi.org/10.1016/j.neuron.2016.09.039>

447 McFarlane, A. C. (2010). The long-term costs of traumatic stress: intertwined physical and  
 448 psychological consequences. *World Psychiatry*, 9(1), 3–10.  
 449 <http://doi.org/10.1002/j.2051-5545.2010.tb00254.x>

450 McLaughlin, K. A., Koenen, K. C., Friedman, M. J., Ruscio, A. M., Karam, E. G., Shahly,  
 451 V., ... & Andrade, L. H. (2015). Subthreshold posttraumatic stress disorder in the  
 452 world health organization world mental health surveys. *Biological Psychiatry*, 77(4),  
 453 375–384. <http://doi.org/10.1016/j.biopsych.2014.03.028>

454 Milad, M. R., Pitman, R. K., Ellis, C. B., Gold, A. L., Shin, L. M., Lasko, ... & Rauch, S. L.  
 455 (2009). Neurobiological basis of failure to recall extinction memory in posttraumatic  
 456 stress disorder. *Biological Psychiatry*, 66(12), 1075–1082.  
 457 <http://doi.org/10.1016/j.biopsych.2009.06.026>

458 National Institute for Health and Care Excellence (2016). *Guideline Scope. Post Traumatic*  
 459 *Stress Disorder Management*. Retrieved from  
 460 <https://www.nice.org.uk/guidance/ng116/documents/final-scope-3>

461 National Institute for Health and Care Excellence (2018). Post-traumatic stress disorder.  
 462 *Guideline NG116*. Retrieved from <https://www.nice.org.uk/guidance/ng116>

- Ness, D., & Calabrese, P. (2016). Stress effects on multiple memory system interactions. *Neural plasticity*, 2016. <http://doi.org/10.1155/2016/4932128>
- Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis. *Psychological Bulletin*, 129(1), 52. <http://dx.doi.org/10.1037/1942-9681.S.1.3>
- Petrie, K., Milligan-Saville, J., Gayed, A., Deady, M., Phelps, A., Dell, L., ... & Harvey, S. B. (2018). Prevalence of PTSD and common mental disorders amongst ambulance personnel: a systematic review and meta-analysis. *Social Psychiatry and Psychiatric Epidemiology*, 1-13. <http://doi.org/10.1007/s00127-018-1539-5>
- Radley, J. J., Sisti, H. M., Hao, J., Rocher, A., McCall, T., Hof, P. R., ... & Morrison, J. H. (2004). Chronic behavioral stress induces apical dendritic reorganization in pyramidal neurons of the medial prefrontal cortex. *Neuroscience*, 125(1), 1-6. <http://doi.org/10.1016/j.neuroscience.2004.01.006>
- Regehr, C., & LeBlanc, V. R. (2017). PTSD, acute stress, performance and decision-making in emergency service workers. *Journal of the American Academy of Psychiatry and the Law*, 45(2), 184-192.
- Rodrigues, S. M., LeDoux, J. E., & Sapolsky, R. M. (2009). The influence of stress hormones on fear circuitry. *Annual Review of Neuroscience*, 32, 289-313. <http://doi.org/10.1146/annurev.neuro.051508.135620>.
- Weathers, F. W., Blake, D. D., Schnurr, P. P., Kaloupek, D. G., Marx, B. P., & Keane, T. M. (2013). The life events checklist for DSM-5 (LEC-5). *Instrument available from the National Center for PTSD at www.ptsd.va.gov*.
- Weiss, D. S. (2007). *The Impact of Event Scale: Revised*, (pp. 219-238). Springer US: Boston, MA. [http://doi.org/10.1007/978-0-387-70990-1\\_10](http://doi.org/10.1007/978-0-387-70990-1_10)
- Yehuda, R., Hoge, C. W., McFarlane, A. C., Vermetten, E., Lanius, R. A., Nievergelt, C. M., ... & Hyman, S. E. (2015). Post-traumatic stress disorder. *Nature Reviews Disease Primers*, 1, 15057. <http://doi.org/10.1038/nrdp.2015.57>